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NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	DEC 01	ChemPort single article sales feature unavailable
NEWS 3	APR 03	CAS coverage of exemplified prophetic substances enhanced
NEWS 4	APR 07	STN is raising the limits on saved answers
NEWS 5	APR 24	CA/CAplus now has more comprehensive patent assignee information
NEWS 6	APR 26	USPATFULL and USPAT2 enhanced with patent assignment/reassignment information
NEWS 7	APR 28	CAS patent authority coverage expanded
NEWS 8	APR 28	ENCOMPLIT/ENCOMPLIT2 search fields enhanced
NEWS 9	APR 28	Limits doubled for structure searching in CAS REGISTRY
NEWS 10	MAY 08	STN Express, Version 8.4, now available
NEWS 11	MAY 11	STN on the Web enhanced
NEWS 12	MAY 11	BEILSTEIN substance information now available on STN Easy
NEWS 13	MAY 14	DGENE, PCTGEN and USGENE enhanced with increased limits for exact sequence match searches and introduction of free HIT display format
NEWS 14	MAY 15	INPADOCDB and INPAFAMDB enhanced with Chinese legal status data
NEWS 15	MAY 28	CAS databases on STN enhanced with NANO super role in records back to 1992
NEWS 16	JUN 01	CAS REGISTRY Source of Registration (SR) searching enhanced on STN
NEWS 17	JUN 26	NUTRACEUT and PHARMAML no longer updated
NEWS 18	JUN 29	IMSCOPROFILE now reloaded monthly
NEWS 19	JUN 29	EPFULL adds Simultaneous Left and Right Truncation (SLART) to AB, MCLM, and TI fields
NEWS 20	JUL 09	PATDPAFULL adds Simultaneous Left and Right Truncation (SLART) to AB, CLM, MCLM, and TI fields
NEWS 21	JUL 14	USGENE enhances coverage of patent sequence location (PSL) data
NEWS 22	JUL 14	CA/CAplus to be enhanced with new citing references features
NEWS 23	JUL 16	GBFULL adds patent backfile data to 1855

NEWS EXPRESS MAY 26 09 CURRENT WINDOWS VERSION IS V8.4,
AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2009.

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FILE 'HOME' ENTERED AT 12:34:58 ON 20 JUL 2009

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=> file caplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
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FILE 'CAPLUS' ENTERED AT 12:35:53 ON 20 JUL 2009
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FILE LAST UPDATED: 19 Jul 2009  (20090719/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Jun 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Jun 2009

CAplus now includes complete International Patent Classification (IPC)
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of databases will soon be updated to include new citing references
information.  This enhancement may impact record import into
database management software.  For additional information, refer
to NEWS 22.

=> PDGFR
          2001 PDGFR
           109 PDGFRS
L1         2016 PDGFR
                (PDGFR OR PDGFRS)

=> antigonist
             8 ANTIGONIST
             6 ANTIGONISTS
L2          14 ANTIGONIST
                (ANTIGONIST OR ANTIGONISTS)

=> antisense
         50772 ANTISENSE
            55 ANTISENSES
L3       50779 ANTISENSE
                (ANTISENSE OR ANTISENSES)
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=> secretory (s) protein
         66214 SECRETORY
             1 SECRETORIES
         66214 SECRETORY
                 (SECRETORY OR SECRETORIES)
       2331670 PROTEIN
       1648432 PROTEINS
       2727050 PROTEIN
                 (PROTEIN OR PROTEINS)
L4       17254 SECRETORY (S) PROTEIN

=> inhibitor
        620889 INHIBITOR
        611342 INHIBITORS
L5      959993 INHIBITOR
                 (INHIBITOR OR INHIBITORS)

=> L1 and L5
L6         926 L1 AND L5

=> L6 an dL3
MISSING OPERATOR L6 AN
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> L3 and L6
L7          17 L3 AND L6

=> L5 and L4
L8        1977 L5 AND L4

=> L1 and L8
L9           1 L1 AND L8

=> L1 and L2
L10          0 L1 AND L2

=> antibody (s) L1
        351175 ANTIBODY
        425467 ANTIBODIES
        560747 ANTIBODY
                 (ANTIBODY OR ANTIBODIES)
L11        125 ANTIBODY (S) L1

=> vector (l) L11
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        133162 VECTORS
        275482 VECTOR
                 (VECTOR OR VECTORS)
L12          2 VECTOR (L) L11

=> D L12 IBIB ABS 1-2

L12  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2009 ACS on STN
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Full Text | More Options

```
ACCESSION NUMBER:          2008:1278701  CAPLUS
DOCUMENT NUMBER:           149:511386
TITLE:                     Antibody fragment scFc and bispecific scFc against
                           PDGFRβ, VEGF-A, HER2/c-erb-2, IL-17A and/or IL-23
```

for treatment of cancer and immune disease
INVENTOR(S): Moore, Margaret D.; Snavely, Marshall D.; Fox, Brian A.; Hoyos, Gabriela H.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 115pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20080260738	A1	20081023	US 2008-106081	20080418
WO 2008131242	A1	20081030	WO 2008-US60852	20080418

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.:		
US 2007-912647P	P	20070418
US 2007-914682P	P	20070427

AB The present invention relates generally to scFc mols. The scFc mols. comprise at least two Fc regions and at least one linker, and can be produced in a variety of single chain configurations. The scFc mols. can further comprise at least one binding entity and/or at least one functional mol. Binding entities can be fused to the scFc mol. in a variety of configurations. The present invention also relates generally to methods for making such mols. and methods for their use. The scFc mols. provided herein can be recombinantly produced. Also provided are monovalent forms of the scFc mols. that have an equiv. or superior ADCC and/or CDC response than do bivalent mols. targeting the same antigens. Provided herein are improved antigen binding compns. Methods for using the scFc mols. of the present inventions are provided.

L12 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | More References

ACCESSION NUMBER: 2002:266311 CAPLUS
DOCUMENT NUMBER: 136:396354
TITLE: Overexpression of ganglioside GM1 results in the dispersion of platelet-derived growth factor receptor from glycolipid-enriched microdomains and in the suppression of cell growth signals
AUTHOR(S): Mitsuda, Teruhiko; Furukawa, Keiko; Fukumoto, Satoshi; Miyazaki, Hiroshi; Urano, Takeshi; Furukawa, Koichi
CORPORATE SOURCE: Department of Biochemistry II, Nagoya University School of Medicine, Nagoya, 466-0065, Japan
SOURCE: Journal of Biological Chemistry (2002), 277(13), 11239-11246
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal
LANGUAGE: English
AB To investigate the mol. mechanisms of gangliosides for the regulation of cell proliferation, Swiss 3T3 cells were transfected with GM2/GD2 synthase and GM1 synthase cDNAs, resulting in the establishment of GM1-expressing (GM1+) lines. Compared with the **vector** control (GM1-) cell lines, GM1+ cells exhibited reduced cell proliferation by stimulation with platelet-derived growth factor (PDGF). In accordance with the reduced cell growth, GM1+ cells showed earlier decreases in the phosphorylation levels of PDGF receptor and less activation of MAP kinases than GM1- cells. To analyze the effects of GM1 expression on the PDGF/PDGF receptor (PDGFR) signals, the glycolipid-enriched microdomain (GEM) was isolated and the following results were obtained. (i) PDGFR predominantly distributed in the non-GEM fraction in GM1+ cells, while it was present in both GEM and non-GEM fractions in GM1- cells. (ii) Activation of **PDGFR** as detected by anti-phosphotyrosine **antibody** occurred almost in parallel with existing amts. of **PDGFR** in each fraction. (iii) GM1 binds with PDGFR in GEM fractions. These findings suggested that GM1 regulates signals via PDGF/PDGFR by controlling the distribution of PDGFR in- and outside of GEM, and also interacting with PDGFR in the GEM fraction as a functional constituent of the microdomain.
REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D L9 TRIB Abs

L9 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Other References

ACCESSION NUMBER: 2006:344369 CAPLUS
DOCUMENT NUMBER: 144:460474
TITLE: A prostate **secretory protein** 94-derived synthetic peptide PCK3145 inhibits VEGF signalling in endothelial cells: implication in tumor angiogenesis
AUTHOR(S): Lamy, Sylvie; Ruiz, Marcia T.; Wisniewski, Jan; Garde, Seema; Rabbani, Shafaat A.; Panchal, Chandra; Wu, Jinzi J.; Annabi, Borhane
CORPORATE SOURCE: Centre de Cancerologie Charles-Bruneau, Hopital Sainte-Justine-UQAM, Montreal, QC, Can.
SOURCE: International Journal of Cancer (2006), 118(9), 2350-2358
CODEN: IJCNAW; ISSN: 0020-7136
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We have previously obsd. that the synthetic peptide corresponding to amino acids 31-45 (PCK3145) of PSP94 can reduce prostate tumor growth in vivo. Moreover, a recently concluded phase IIa clin. trial with patients with hormone refractory prostate cancer indicated that PCK3145 down-regulates the levels of plasma matrix metalloproteinase (MMP)-9, a MMP involved in metastasis and tumor angiogenesis. The purpose of our study was to investigate the mol. mechanisms of action of PCK3145 and whether this peptide could antagonize tumor neovascularization. We show that, in a syngeneic in vivo model of rat prostate cancer, the expression of endothelial cell (EC) specific CD31, a marker of tumor vessel d., was decreased by 43% in PCK3145-treated animals. In vitro, PCK3145 specifically antagonized in a dose-dependent manner the VEGF-induced ERK phosphorylation as well as the phosphorylation of the VEGFR-2 in cultured EC (HUVEC). These anti-VEGF effects were partly reproduced by pharmacol.

inhibitors such as PD98059 and PTK787, suggesting that PCK3145 inhibits the tyrosine kinase activity assocd. to VEGFR-2, which in turn prevents intracellular signaling through the MAPK cascade. Moreover, PCK3145 was also found to inhibit the PDGF-induced phosphorylation of **PDGFR** in smooth muscle cells. Finally, PCK3145 inhibited in vitro EC tubulogenesis and VEGF-induced MMP-2 secretion suggesting its potential implication as an antiangiogenic agent. Our study demonstrates that PCK3145 interferes with the tyrosine kinase activity assocd. with VEGF signaling axis in EC. The antiangiogenic properties of this peptide could be highly beneficial and exploited in novel antiangiogenic therapies, for patients with various cancers.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D L7 IBIB ABS 1-17

L7 ANSWER 1 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | More Patents

ACCESSION NUMBER: 2009:456612 CAPLUS
DOCUMENT NUMBER: 150:414291
TITLE: Methods of treatment of opioid tolerance, physical dependence, pain, and addiction with **inhibitors** of certain growth factor receptors
INVENTOR(S): Gutstein, Howard B.
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System, USA
SOURCE: PCT Int. Appl., 34pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2009048947	A1	20090416	WO 2008-US79198	20081008

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2007-978641P P 20071009

AB Methods of preventing the development and reversing or partially reversing opioid tolerance in a subject are provided herein. Such methods include the step of administering to a subject in need thereof a therapeutically effective amt. of a **PDGFR** modulator or EGFR modulator alone or together with an opiate analgesic. The growth factor receptor modulators are administered spinally, i.v., i.p., i.m., s.c. or orally. The methods can also be used for the treatment of refractory neuropathic pain, phys. dependence or addiction.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2009:139541 CAPLUS
DOCUMENT NUMBER: 150:191158
TITLE: Combinations of MEK **inhibitors** and Raf kinase **inhibitors** and uses thereof
INVENTOR(S): Miner, Jeffrey N.; Chapman, Mark S.; Quart, Barry; Adjei, Alex; Yu, Chunrong
PATENT ASSIGNEE(S): Ardea BioSciences, Inc., USA
SOURCE: PCT Int. Appl., 148pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2009018238	A1	20090205	WO 2008-US71397	20080728
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
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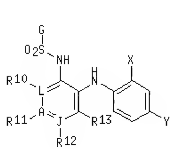
US 20080058340	A1	20080306	US 2007-830733	20070730
US 20080255133	A1	20081016	US 2008-16897	20080118

PRIORITY APPLN. INFO.:

US 2007-830733	A	20070730
US 2008-16897	A	20080118
US 2005-701814P	P	20050721
US 2005-706719P	P	20050808
US 2005-731633P	P	20051028
WO 2006-US28326	A2	20060721
US 2006-833886P	P	20060728
US 2007-885849P	P	20070119

OTHER SOURCE(S): CASREACT 150:191158; MARPAT 150:191158
GI



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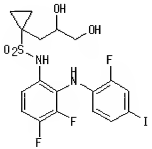

II

AB This invention concerns combinations of **inhibitors** of MEK, Raf protein kinases, and other kinases including VEGFR1-3 and **PDGFR**-β. This invention also concerns pharmaceutical compns. comprising the compds. I [G = 1-(2,3-dihydroxypropyl)cyclopropyl, cyclopropyl, etc.; R10 = H, halo, CN, etc.; R11 = (un)substituted 5-6 membered heterocyclic contg. 1-5 heteroatoms selected from O, N and S; R12 = H, halo, F, O; R13 = H, halo, OH, etc.; X, Y = F, I, Br, alkyl, etc.; A, J, L = C, CH, NH, N, O, N(Me)] and methods of use of the compds. I and compns. described herein, including the use in the treatment and/or prevention of cancer and other hyperproliferative disorders. For example, a multi-step synthesis of II, starting from cyclopropanesulfonyl chloride, was given. Compds. I were tested in various biol. assays, alone or in combination with other therapeutic agents (data given).

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Other References

ACCESSION NUMBER: 2008:859686 CAPLUS
DOCUMENT NUMBER: 149:167943
TITLE: Methods and compositions for treating cancer using Bcl-2 **antisense** oligomers, tyrosine kinase **inhibitors**, and chemotherapeutic agents
INVENTOR(S): Brown, Bob D.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 11pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20080171718	A1	20080717	US 2007-935654	20071106
WO 2008058225	A2	20080515	WO 2007-US84014	20071108
WO 2008058225	A3	20080904		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.:		
US 2006-864859P	P	20061108
US 2007-935654	A	20071106

AB Methods and compns. are provided for treating cell proliferation-related disorders, e.g. cancer. Methods of inhibiting the growth of cancer cells comprise contacting the cancer cells with a Bcl-2 **antisense** oligomer; contacting the cancer cells with a tyrosine kinase **inhibitor**; and contacting the cancer cells with a cytotoxic chemotherapeutic agent. Methods of treating cancer in a human comprise administering to the human a Bcl-2 **antisense** oligomer, a tyrosine kinase **inhibitor**, and a cytotoxic chemotherapeutic agent. Kits contg. compns. in amts. sufficient

for at least one cycle of treatment comprise a triplet combination therapy of a Bcl-2 **antisense** oligomer, a tyrosine kinase **inhibitor**, and a cytotoxic chemotherapeutic agent. In selected embodiments, the tyrosine kinase **inhibitor** is one that targets cell surface kinase receptors, such as VEGFR (e.g., VEGFR1, VEGFR2, VEGFR3), **PDGFR**, KIT, and FLT-3.

L7 ANSWER 4 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | More References

ACCESSION NUMBER: 2008:583352 CAPLUS
DOCUMENT NUMBER: 148:529452
TITLE: Methods and compositions for treating cancer using Bcl-2 **antisense** oligomers, tyrosine kinase **inhibitors**, and chemotherapeutic agents
INVENTOR(S): Brown, Bob D.
PATENT ASSIGNEE(S): Genta Inc., USA
SOURCE: PCT Int. Appl., 22 pp., which
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2008058225	A2	20080515	WO 2007-US84014		20071108
WO 2008058225	A3	20080904			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
US 20080171718	A1	20080717	US 2007-935654		20071106
PRIORITY APPLN. INFO.:			US 2006-864859P	P	20061108
			US 2007-935654	A	20071106

AB Methods and compns. are provided for treating cell proliferation-related disorders, e.g. cancer. Methods of inhibiting the growth of cancer cells comprise contacting the cancer cells with a Bcl-2 **antisense** oligomer; contacting the cancer cells with a tyrosine kinase **inhibitor**; and contacting the cancer cells with a cytotoxic chemotherapeutic agent. Methods of treating cancer in a human comprise administering to the human a Bcl-2 **antisense** oligomer, a tyrosine kinase **inhibitor**, and a cytotoxic chemotherapeutic agent. Kits contg. compns. in amts. sufficient for at least one cycle of treatment comprise a triplet combination therapy of a Bcl-2 **antisense** oligomer, a tyrosine kinase **inhibitor**, and a cytotoxic chemotherapeutic agent. In selected embodiments, the tyrosine kinase **inhibitor** is one that targets cell surface kinase receptors, such as VEGFR (e.g., VEGFR1, VEGFR2, VEGFR3), **PDGFR**, KIT, and FLT-3.

L7 ANSWER 5 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | More References

ACCESSION NUMBER: 2007:1454433 CAPLUS

DOCUMENT NUMBER: 148:85569
TITLE: Pan-cell surface receptor (HER family)-specific therapeutic multimers interacting with at least two different receptor ligands
INVENTOR(S): Shepard, H. Michael; Jin, Pei; Burton, Louis E.; Beryt, Malgorzata
PATENT ASSIGNEE(S): Receptor Biologix Inc., USA
SOURCE: PCT Int. Appl., 320pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007146959	A2	20071221	WO 2007-US71041	20070612
WO 2007146959	A3	20080724		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			
AU 2007257683	A1	20071221	AU 2007-257683	20070612
CA 2655205	A1	20071221	CA 2007-2655205	20070612
EP 2044115	A2	20090408	EP 2007-784419	20070612
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS				
IN 2009CN00146	A	20090529	IN 2009-CN146	20090109
KR 2009031897	A	20090330	KR 2009-700615	20090112

PRIORITY APPLN. INFO.:

US 2006-813260P	P	20060612
US 2006-848542P	P	20060929
US 2007-878941P	P	20070105
WO 2007-US71041	W	20070612

AB Provided are pan-cell surface receptor-specific therapeutics, methods for prepg. them and methods of treatment using them. Among the pan-cell surface receptor-specific therapeutics are pan-HER (ErbB, EGFR) family-specific therapeutics that interact with at least two different HER receptor ligands and/or dimerize with or interact with two or more HER cell surface receptors. Her family of receptors includes epidermal growth factor receptor (HER1), neu receptor (HER2), and neuregulin receptors (HER3 and HER4). By virtue of these properties, the therapeutics modulate the activity of at least two cell surface receptors and are useful for therapeutic purposes. Provided herein are multimers of an extracellular domain (ECD) of two cell surface receptors, including heteromultimers that contain modified ECDs. For example, EGFR1, which is activated by EGF and generally is not stimulated by NRG-2β, was modified so that both ligands interact with the EGFR ECD to promote receptor dimerization/receptor signaling. The therapeutic chimeric protein of the invention may also include ECD of IGF1-R, VEGFR, FGFR, TNFR, PDGFR, MET, Tie, RAGE, EPH receptor and T cell receptor.

L7 ANSWER 6 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | More References

ACCESSION NUMBER: 2007:1243268 CAPLUS
DOCUMENT NUMBER: 147:480419
TITLE: Methods and compositions for modulation of blood-neural barrier for treatment of CNS and other disorders
INVENTOR(S): Eriksson, Ulf; Lawrence, Daniel; Su, Enming Joe; Strickland, Dudley; Yeppes, Manuel; Fredriksson, Linda
PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, USA
SOURCE: PCT Int. Appl., 75pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007124308	A2	20071101	WO 2007-US66804	20070417
WO 2007124308	A3	20080221		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			
AU 2007240429	A1	20071101	AU 2007-240429	20070417
US 20070265203	A1	20071115	US 2007-736499	20070417
EP 2021028	A2	20090211	EP 2007-797242	20070417
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS				

PRIORITY APPLN. INFO.:		
US 2006-792318P	P	20060417
US 2006-828506P	P	20061006
WO 2007-US66804	W	20070417

AB Methods and compns. for modulating blood-neural barrier (BNB) for the treatment of CNS conditions such as edema, and for increased drug delivery efficacy across the BNB are provided. The present invention further relates to improved tPA treatment of ischemic cerebrovascular and related diseases in combination with antagonism of the PDGF signaling pathway. The inventive method and compn. is particularly suitable for conjunctive therapy of ischemic stroke using tPA and an anti-PDGF-C antagonist or an anti-**PDGFR**-α antagonist. Thus, tPA, as well as PDGF-CC in the cerebrospinal fluid, were potent inducers of opening of the blood-brain barrier (BBB). However, tPA together with PDGF-CC did not significantly increase BBB opening, suggesting that both tPA and PDGF-CC were able to open the BBB, but the effects were not synergistic or additive.

L7 ANSWER 7 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | More References

ACCESSION NUMBER: 2006:884327 CAPLUS

DOCUMENT NUMBER: 145:262978
TITLE: Involvement of insulin-like growth factor type 1 receptor and protein kinase Cδ in Bis(maltolato)oxovanadium(IV)-induced phosphorylation of protein kinase B in hepG2 cells
AUTHOR(S): Mehdi, Mohamad Z.; Vardatsikos, George; Pandey, Sanjay K.; Srivastava, Ashok K.
CORPORATE SOURCE: Laboratory of Cell Signaling, Montreal Diabetes Research Center, Centre hospitalier de l'Universite de Montreal, Universite de Montreal, Montreal, QC, H1W 4A4, Can.
SOURCE: Biochemistry (2006), 45(38), 11605-11615
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Vanadium(IV) oxo-bis(maltolato) (BMOV), an organo-vanadium compd., is a potent insulinomimetic agent and improves glucose homeostasis in various models of diabetes. We have shown previously that BMOV stimulates the phosphorylation of PKB which may contribute as one of the mechanisms for the insulinomimetic effect of this compd. However, the upstream mechanism of BMOV-induced PKB phosphorylation remains elusive. Therefore, in this study, we examine the upstream events leading to BMOV-induced PKB phosphorylation in HepG2 cells. Since BMOV is an **inhibitor** of protein tyrosine phosphatases and through enhanced tyrosine phosphorylation may activate various protein tyrosine kinases (PTK), we have investigated the potential role of different receptor or nonreceptor PTK in mediating BMOV-induced PKB phosphorylation. Among several pharmacol. **inhibitors** that were tested, only AG1024, a selective **inhibitor** of IGF-1R-PTK, almost completely blocked BMOV-stimulated phosphorylation of PKB. In contrast, AG1295 and AG1478, specific **inhibitors** of **PDGFR** and EGFR, resp., were unable to block the BMOV response. Moreover, efficient redn. of the level of IGF-1R protein expression by **antisense** oligonucleotides (ASO) attenuated BMOV-induced PKB phosphorylation. BMOV-induced PKB phosphorylation was assocd. with an increased level of tyrosine phosphorylation of the IRβ subunit, IGF-1Rβ subunit, IRS-1, and p85α subunit of PI3-kinase. However, this response was independent of IR-PTK activity because in cells overexpressing a PTK-inactive form of IR, insulin response was attenuated while the effect of BMOV remained intact. A role of PKC in BMOV-induced response was also tested. Pharmacol. inhibition with chelerythrine, a nonselective PKC **inhibitor**, or rottlerin, a PKCδ **inhibitor**, as well as chronic treatment with PMA attenuated BMOV-induced PKB phosphorylation. In contrast, Go6976 and RO31-8220 PKCα/β selective **inhibitors** failed to alter the BMOV effect. Taken together, these data suggest that IGF-1R and PKCδ are required to stimulate PKB phosphorylation in response to BMOV in HepG2 cells and provide new insights into the mol. mechanism by which this compd. exerts its insulinomimetic effects.

REFERENCE COUNT: 71 THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 8 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Web References

ACCESSION NUMBER: 2006:636808 CAPLUS
DOCUMENT NUMBER: 145:89828
TITLE: Method for treating diseases associated with abnormal kinase activity
INVENTOR(S): Lyons, John; Rubinfeld, Joseph
PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 18 pp., Cont.-in-part of U.S. Ser. No. 206,854.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20060140947	A1	20060629	US 2005-181368	20050713
US 20030147813	A1	20030807	US 2002-71849	20020207
US 20040127453	A1	20040701	US 2002-206854	20020726
US 6998391	B2	20060214		
PRIORITY APPLN. INFO.:			US 2002-71849	A2 20020207
			US 2002-206854	A2 20020726

AB Methods are provided for treating diseases assocd. with abnormal activity of kinases. The method comprises: administering a DNA methylation **inhibitor** to the patient in therapeutically effective amt.; and administering a kinase **inhibitor** to the patient in therapeutically effective amt., such that the in vivo activity of the kinase is reduced relative to that prior to the treatment. The method can be used to treat cancer assocd. with abnormal activity of kinases such as phosphatidylinositol 3'-kinase (PI3K), protein kinases including serine/threonine kinases such as Raf kinases, protein kinase kinases such as MEK, and tyrosine kinases such as those in the epidermal growth factor receptor family (EGFR), platelet-derived growth factor receptor family (**PDGFR**), vascular endothelial growth factor receptor (VEGFR) family, nerve growth factor receptor family (NGFR), fibroblast growth factor receptor family (FGFR) insulin receptor family, ephrin receptor family, Met family, Ror family, c-kit family, Src family, Fes family, JAK family, Fak family, Btk family, Syk/ZAP-70 family, and Abl family.

L7 ANSWER 9 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | String References

ACCESSION NUMBER: 2006:99983 CAPLUS
DOCUMENT NUMBER: 144:184708
TITLE: Use of K-252a and kinase **inhibitors** for the prevention or treatment of HMGB1-associated pathologies
INVENTOR(S): Fumero, Silvano; Pilato, Francesco, P.; Barone, Domenico; Bertarione, Rava, Rossa, Luisa; Mainero, Valentina; Traversa, Silvio
PATENT ASSIGNEE(S): Creabilis Therapeutics S.p.A., Italy; Bio3research Srl
SOURCE: PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006010628	A1	20060202	WO 2005-EP8258	20050729

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK,

SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

AU 2005266447	A1	20060202	AU 2005-266447		20050729
CA 2575272	A1	20060202	CA 2005-2575272		20050729
EP 1771178	A1	20070411	EP 2005-778429		20050729
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR					
JP 2008508220	T	20080321	JP 2007-523023		20050729
MX 2007001155	A	20070814	MX 2007-1155		20070129
US 20080317809	A1	20081225	US 2007-658701		20070129
PRIORITY APPLN. INFO.:			US 2004-591880P	P	20040729
			US 2005-647007P	P	20050127
			WO 2005-US8258	W	20050311
			WO 2005-EP8258	W	20050729

AB The present invention relates to the use of K-252a, a physiol. active substance produced by microorganisms, and/or a kinase **inhibitor** and of its salts or synthetic and/or chem. modified derivs. for the prevention or treatment of HMGB1-assocd. pathologies. More particularly, the present invention relates to the use of K-252a for the prevention or treatment of restenosis.

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 10 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Other References

ACCESSION NUMBER: 2006:29606 CAPLUS
DOCUMENT NUMBER: 144:121754
TITLE: Gene expression profile for predicting activity of compounds that interact with and/or modulate protein tyrosine kinases and/or protein tyrosine pathways in lung cancer cells
INVENTOR(S): Huang, Fei; Reeves, Karen A.; Han, Xia; Fairchild, Craig R.; Shaw, Peter
PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA
SOURCE: PCT Int. Appl., 130 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006005035	A2	20060112	WO 2005-US23687	20050629
WO 2006005035	A3	20090409		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF,				

CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

US 20060019284 A1 20060126 US 2005-169041 20050628
EP 1766080 A2 20070328 EP 2005-769088 20050629
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU
JP 2008504843 T 20080221 JP 2007-520422 20050629
PRIORITY APPLN. INFO.: US 2004-584405P P 20040630
WO 2005-US23687 W 20050629

AB The present invention describes polynucleotides that have been discovered to correlate to the relative intrinsic sensitivity or resistance of cells, e.g., lung cell lines, to treatment with compds. that interact with and modulate, e.g., inhibit, protein tyrosine kinases, such as, for example, members of the Src family of tyrosine kinases, e.g., Src, Fgr, Fyn, Yes, Blk, Hck, Lck and Lyn, as well as other protein tyrosine kinases, including, Bcr-abl, Jak, **PDGFR**, c-kit and Ephr. These polynucleotides have been shown, through a weighted voting cross validation program, to have utility in predicting the resistance and sensitivity of lung cell lines to the compds. The expression level of some polynucleotides is regulated by treatment with a particular protein tyrosine kinase **inhibitor** compd., thus indicating that these polynucleotides are involved in the protein tyrosine kinase signal transduction pathway, e.g., Src tyrosine kinase. The Affymetrix human HG-U133 GeneChip set of over 44,792 probe sets was used to identify 129 polynucleotides that are highly correlated with a resistance/sensitivity phenotype classification of 23 lung cell lines subjected to treatment with the protein tyrosine kinase **inhibitor** compd. BMS-A. Of the 129 predictor polynucleotides, 81 polynucleotides highly expressed in the cell lines were classified as sensitive to BMS-A, while 48 polynucleotides highly expressed in the cell lines were classified as resistant to BMS-A. Such polynucleotides, whose expression levels correlate highly with drug sensitivity or resistance and which are modulated by treatment with the compds., comprise polynucleotide predictor or marker sets useful in methods of predicting drug response, and as prognostic or diagnostic indicators in disease management, particularly in those disease areas, e.g., lung cancer, in which signaling through the protein tyrosine kinase pathway, such as the Src tyrosine kinase pathway, is involved with the disease process.

L7 ANSWER 11 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | CHIPS | References

ACCESSION NUMBER: 2005:1021645 CAPLUS
DOCUMENT NUMBER: 143:279373
TITLE: Method of inhibiting tumor proliferation
INVENTOR(S): Sueishi, Katsuo; Yonemitsu, Yoshikazu; Shikada, Yasunori; Tsutsumi, Norifumi; Hasegawa, Mamoru
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005087269	A1	20050922	WO 2005-JP4485	20050315

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2560046	A1	20050922	CA 2005-2560046		20050315
US 20080199438	A1	20080821	US 2007-598947		20070327
PRIORITY APPLN. INFO.:			JP 2004-74570	A	20040316
			WO 2005-JP4485	W	20050315

AB It is intended to provide a method of inhibiting tumor proliferation which comprises the step of inhibiting the expression of PDGF-A or the binding of PDGF-A homodimer to **PDGFR**α. Activation of the **PDGFR** α-p70S6K signal transduction pathway by PDGF-AA, which is an important factor in tumor angiogenesis, relates to the prognosis of a patient suffering from tumor. By inhibiting the expression of PDGF-A in a tumor or a tissue around it or by inhibiting the binding of PDGF-A homodimer to **PDGFR**α, angiogenesis in the tumor and retention of the blood vessels can be inhibited and thus the tumor proliferation can be inhibited.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 12 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Other References

ACCESSION NUMBER: 2004:533967 CAPLUS
DOCUMENT NUMBER: 141:65147
TITLE: Method for treating diseases associated with abnormal tyrosine kinase activity by administering a DNA methylation **inhibitor** and a tyrosine kinase **inhibitor**
INVENTOR(S): Lyons, John; Rubinfeld, Joseph
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 19 pp., Cont.-in-part of U.S. Ser. No. 71,849.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20040127453	A1	20040701	US 2002-206854	20020726
US 6998391	B2	20060214		
US 20030147813	A1	20030807	US 2002-71849	20020207
CA 2474174	A1	20030814	CA 2003-2474174	20030206
WO 2003065995	A2	20030814	WO 2003-US3537	20030206
WO 2003065995	A3	20051013		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,

KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2003215065	A1	20030902	AU 2003-215065	20030206
EP 1572075	A2	20050914	EP 2003-710881	20030206
EP 1572075	A3	20051207		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

US 20060140947	A1	20060629	US 2005-181368	20050713
PRIORITY APPLN. INFO.:			US 2002-71849	A2 20020207
			US 2002-206854	A 20020726
			WO 2003-US3537	W 20030206

AB Methods are provided for treating diseases assocd. with abnormal activity of kinases. The method comprises: administering a DNA methylation **inhibitor** to the patient in therapeutically effective amt.; and administering a kinase **inhibitor** to the patient in therapeutically effective amt., such that the in vivo activity of the kinase is reduced relative to that prior to the treatment. The method can be used to treat cancer assocd. with abnormal activity of kinases such as phosphatidylinositol 3'-kinase (PI3K), protein kinases including serine/threonine kinases such as Raf kinases, protein kinase kinases such as MEK, and tyrosine kinases such as those in the epidermal growth factor receptor family (EGFR), platelet-derived growth factor receptor family (**PDGFR**), vascular endothelial growth factor receptor (VEGFR) family, nerve growth factor receptor family (NGFR), fibroblast growth factor receptor family (FGFR) insulin receptor family, ephrin receptor family, Met family, Ror family, c-kit family, Src family, Fes family, JAK family, Fak family, Btk family, Syk/ZAP-70 family, and Abl family.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 13 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | More References

ACCESSION NUMBER: 2004:203933 CAPLUS
DOCUMENT NUMBER: 140:247003
TITLE: Expressed polynucleotides markers for predicting activity of compounds that interact with and/or modulate protein tyrosine kinases and/or protein tyrosine kinase pathways in breast cells
INVENTOR(S): Huang, Fei; Han, Xia; Reeves, Karen A.; Amler, Lucas; Fairchild, Craig R.; Lee, Francis Y.; Shaw, Peter
PATENT ASSIGNEE(S): Bristol-Myers Squibb Co., USA
SOURCE: PCT Int. Appl., 649 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004020583	A2	20040311	WO 2003-US26491	20030826
WO 2004020583	A3	20060302		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SZ, BE, CY,

FR, GR, IE, IT, MC, NL, SI, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2003278725	A1	20040319	AU 2003-278725		20030826
EP 1572957	A2	20050914	EP 2003-770252		20030826
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
JP 2006515742	T	20060608	JP 2004-532963		20030826
PRIORITY APPLN. INFO.:			US 2002-406385P	P	20020827
			WO 2003-US26491	W	20030826

AB The present invention describes polynucleotides that have been discovered to correlate to the relative intrinsic sensitivity or resistance of cells (e.g., breast cell lines) to treatment with compds. that interact with and modulate (e.g., inhibit) protein tyrosine kinases, such as, for example, members of the Src family of tyrosine kinases (e.g., Src, Fgr, Fyn, Yes, Blk, Hck, Lck and Lyn), as well as other protein tyrosine kinases, including, Bcr-abl, Jak, **PDGFR**, c-kit and Eph receptors. These polynucleotides have been shown, through a weighted voting cross validation program, to have utility in predicting the resistance and sensitivity of breast cell lines to the compds. Thus, 137 polynucleotides are provided that highly correlate with a resistance/sensitivity phenotype classification of 23 breast cell lines for the protein tyrosine kinase **inhibitor** BMS-A. The expression level or phosphorylation status of some polynucleotides is regulated by treatment with a particular protein tyrosine kinase **inhibitor** compd., thus indicating that these polynucleotides are involved in the protein tyrosine kinase signal transduction pathway. Such polynucleotides, whose expression levels correlate highly with drug sensitivity or resistance and which are modulated by treatment with the compds., comprise polynucleotide predictor or marker sets useful in methods of predicting drug response, and as prognostic or diagnostic indicators in disease management, particularly in those disease areas, e.g., breast cancer, in which signaling through the protein tyrosine kinase pathway is involved with the disease process.

L7 ANSWER 14 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Other References

ACCESSION NUMBER:	2003:633416 CAPLUS
DOCUMENT NUMBER:	139:173786
TITLE:	Method for treating diseases associated with abnormal kinase activity
INVENTOR(S):	Lyons, John; Rubinfeld, Joseph
PATENT ASSIGNEE(S):	Supergen, Inc., USA
SOURCE:	PCT Int. Appl., 64 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003065995	A2	20030814	WO 2003-US3537	20030206
WO 2003065995	A3	20051013		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,				

LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 20030147813	A1	20030807	US 2002-71849		20020207
US 20040127453	A1	20040701	US 2002-206854		20020726
US 6998391	B2	20060214			
CA 2474174	A1	20030814	CA 2003-2474174		20030206
AU 2003215065	A1	20030902	AU 2003-215065		20030206
EP 1572075	A2	20050914	EP 2003-710881		20030206
EP 1572075	A3	20051207			

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

PRIORITY APPLN. INFO.:	US 2002-71849	A1	20020207
	US 2002-206854	A1	20020726
	WO 2003-US3537	W	20030206

AB Methods are provided for treating diseases assocd. with abnormal activity of kinases such as chronic myelogenous leukemia. The method comprises: administering a DNA methylation **inhibitor** to the patient in therapeutically effective amt.; and administering a kinase **inhibitor** such as imatinib mesylate to the patient in therapeutically effective amt., such that the in vivo activity of the kinase is reduced relative to that prior to the treatment. The method can be used to treat cancer assocd. with abnormal activity of kinases such as phosphatidylinositol 3'-kinase (PI3K), protein kinases including serine/threonine kinases such as Raf kinases, protein kinase kinases such as MEK, and tyrosine kinases such as those in the epidermal growth factor receptor family (EGFR), platelet-derived growth factor receptor family (**PDGFR**), vascular endothelial growth factor receptor (VEGFR) family, nerve growth factor receptor family (NGFR), fibroblast growth factor receptor family (FGFR) insulin receptor family, ephrin receptor family, Met family, Ror family, c-kit family, Src family, Fes family, JAK family, Fak family, Btk family, Syk/ZAP-70 family, and Abl family.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 15 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Citing References

ACCESSION NUMBER:	2003:475720 CAPLUS
DOCUMENT NUMBER:	140:70616
TITLE:	Effect of **PDGFR**-β **antisense** oligonucleotides on proliferation of cultured rat aortic vascular smooth muscle cells
AUTHOR(S):	Gu, Chunhu; Hou, Yingping; Qiao, Hongqing; Li, Yan; Wang, Yunya; Lin, Guocheng
CORPORATE SOURCE:	Xijing Hospital, Fourth Military Medical University, Xian, Shanxi Province, 710033, Peop. Rep. China
SOURCE:	Disi Junyi Daxue Xuebao (2002), 23(7), 589-592 CODEN: DJDXEG; ISSN: 1000-2790
PUBLISHER:	Disi Junyi Daxue Xuebao Bianjibu
DOCUMENT TYPE:	Journal
LANGUAGE:	Chinese

AB The effect of platelet-derived growth factor receptor-β (**PDGFR**-β) **antisense** oligonucleotides (AODN) on the proliferation of cultured rat aortic vascular smooth muscle cells (VSMC) was studied. The cultured rat aortic VSMC model was established in vitro, then the

cells were divided into **antisense** oligonucleotide (AODN) group, sense oligonucleotide (SODN) group, scrambled oligonucleotide (CODN) group, and control group. The cells in AODN group were subdivided into five small groups by the AODN concn. of 1, 2.5, 5, 10, 15 μmol/L-1. MTT assay, flow cytometry, immunohistochem. for the proliferating cell nuclear antigene (PCNA) were used to det. the effects of **PDGFR**-β AODN on the proliferation of VSMC. The percentage of quiescent cells (G0/G1) of AODN group at 48 h (0.70) was much higher than that of control group (0.07), P<0.05. The percentages of PCNA expression with 5-15 μmol/L-1 AODN were between 6.4% and 20.4%, which were lower than that of control group (73.8%), SODN group (73.9%), and CODN group (75.6%) (P<0.05). The inhibiting rate of the proliferation of VSMC in 10 μmol/L-1 AODN group at 48 h (64.7%) was higher than that of SODN group (8.1%), CODN group (11.8%), 5 μmol/L-1 AODN group (45.4%), and 10 μmol/L-1 AODN group at 24 h (9.1%) (P<0.05). **PDGFR**-β AODN could inhibit the proliferation of cultured VSMC in a dose- and time-dependent pattern.

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Full Text | Other References

ACCESSION NUMBER: 2002:794308 CAPLUS
DOCUMENT NUMBER: 137:316046
TITLE: Localized oligonucleotide therapy for preventing restenosis
INVENTOR(S): Sirois, Martin G.; Edelman, Elazer R.; Rosenberg, Robert D.; Simons, Michael
PATENT ASSIGNEE(S): Can.
SOURCE: U.S. Pat. Appl. Publ., 35 pp., Cont.-in-part of U. S. Ser. No. 241,561, abandoned.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 20020151513	A1	20021017	US 2001-945131		20010831
CA 2228977	A1	19990507	CA 1998-2228977		19980203
PRIORITY APPLN. INFO.:			CA 1998-2228977	A	19980203
			US 1999-241561	B2	19990201
			CA 1997-2215360	A	19971107

AB **Antisense** oligonucleotide gene therapy selective for the 5' region of **PDGFR**-β subunit mRNA was used in attempt to prevent intimal thickening following rat carotid arterial injury. Sustained perivascular application of the **antisense** oligomers for 14 days reduced **PDGFR**-β protein overexpression and prevented neointima formation by 80%. Alternatively, a bolus of **antisense** oligomers reduced the **PDGFR**-β protein expression by at least 90% for at least 28 days. Specificity was verified by the absence of effects on the expression of a non-targeted gene **PDGFR**-α. These data demonstrated that **antisense** oligonucleotide sequences can effectively suppress a growth factor receptor, and the redn. of intimal hyperplasia after injury correlates with the extent to which these oligomers inhibited **PDGFR**-β protein expression. Advantageously, redn. of intimal hyperplasia was also accomplished with an almost completely restored endothelial function. Methods and materials useful for preventing restenosis are described and claimed.

L7 ANSWER 17 OF 17 CAPLUS COPYRIGHT 2009 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1999:504155 CAPLUS
DOCUMENT NUMBER: 131:255617
TITLE: Reduced receptor expression for platelet-derived growth factor and epidermal growth factor in dividing mouse lung epithelial cells
AUTHOR(S): Rice, Pamela L.; Porter, Stephanie E.; Koski, Kelli M.; Ramakrishna, Gayatri; Chen, Aaron; Schrump, David; Kazlauskas, Andrius; Malkinson, Alvin M.
CORPORATE SOURCE: Department of Pharmaceutical Sciences, School of Pharmacy, University of Colorado Health Sciences Center, Denver, CO, 80262, USA
SOURCE: Molecular Carcinogenesis (1999), 25(4), 285-294
CODEN: MOCAE8; ISSN: 0899-1987
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The roles of growth factors in mouse lung neoplasia were investigated by examg. receptors for platelet-derived growth factor (PDGF) and epidermal growth factor (EGF) in epithelial cell lines. Whereas nontumorigenic lung cells expressed mRNA and protein for PDGF receptor (**PDGFR**)-α, **PDGFR**-β, and EGF receptor (EGFR), five of six neoplastic lines did not. Because this exceptional tumorigenic cell line grows slowly, we hypothesized that receptor levels increased with cell stasis. To test this hypothesis, serum concns. were manipulated, and log-phase and post-confluent cells were compared. Consistent with our hypothesis, **PDGFR**-α and EGFR contents, but not **PDGFR**-β contents, increased at stasis. Ki-ras mutation initiates lung tumorigenesis in mice, but activation of Ki-ras did not affect receptor expression. This was detd. both by transfecting nontumorigenic cells with activated Ki-ras and neoplastic cells with a Ki-ras **antisense** construct and by diminishing Ki-ras activation by using a farnesyltransferase **inhibitor**. Stasis-assocd. upregulation of growth-factor receptor expression suggests a function in lung cell differentiation that is abrogated during neoplastic growth.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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